Emotional numbing in posttraumatic stress disorder: current and future research directions

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Objective: Despite being understudied and poorly understood relative to the chronic fear, anxiety and other aversive emotional states that occur in the immediate aftermath of trauma, emotional numbing has become a core defining feature of posttraumatic stress disorder (PTSD).

Method: This paper seeks to briefly review the literature bearing on these seemingly disparate emotional responses to trauma as well as theoretical accounts of emotional numbing that have been proffered to date. We then offer an alternative theory of post-traumatic emotional functioning and review empirical support for this model.

Result: The experience of trauma produces very intense emotions such as overwhelming fear, horror, and anxiety, and these reactions can linger for a lifetime. Many trauma survivors also report restrictions in their emotional experience – a phenomenon most commonly referred to as emotional numbing. In contrast to previous accounts of posttraumatic emotional functioning our model posits that individuals with PTSD have difficulty expressing positive emotions as a result of re-experiencing states. We further argue that patients with PTSD are capable of experiencing and expressing the full range of emotions that were available pretraumatically.

Conclusion: Our model holds that individuals with PTSD are not, in fact, 'emotionally numb' as a result of traumatic experience. Rather, PTSD is associated with hyperresponsivity to negatively valenced emotional stimuli. Consequently, patients with PTSD require more intense positive stimulation to access the full complement of appetitive or pleasant emotional behaviour.

Key words: emotional numbing, posttraumatic stress disorder, PTSD.

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Clinicians and researchers from all theoretical perspectives concur that a defining feature of trauma is the elicitation of intense basic emotions – typically such intense fear that terms like terror and horror are more appropriate descriptors [1]. Extreme fear accompanied by very high arousal is not the only emotional response during trauma. Anguish, dread, disgust, rage, sorrow, and shame are also common descriptors of the emotional experience of trauma [2,3]. Individuals also report experiences during, and especially immediately after trauma that reflect unresponsiveness to stimulation, a lack of emotional involvement in their surroundings, shock, and exhaustion [4,5]. The hallmark symptoms of PTSD parallel the divergent emotion-related experiences of acute trauma: an apparently shifting pattern of intense emotionality triggered by reminders of trauma as well as

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emotional response deficits or what has been termed 'emotional numbing'. Disrupted emotional experiences are central defining characteristics of the PTSD syndrome [6]. Individuals with PTSD report intense negative affect and physiological arousal when reminded of their trauma. On the other hand, individuals with PTSD also report restrictions in their capacity to feel, or emotional numbing [6]. Emotional numbing has also been described as 'psychic numbing', or 'emotional anaesthesia', and is further described as involving a complaint of a loss of 'the ability to become interested in previously enjoyed activities', or 'the ability to feel emotions of any type, especially those associated with intimacy, tenderness, and sexuality' [6]. Although several researchers have argued that these two types of experiences are phasically related [7–9], the relationship between these severely contrasting response classes has not been sufficiently specified nor researched.

Emotional numbing: a need for definitional clarity

In contrast to advances in understanding emotional arousal and conditioned negative affect in PTSD, the emotional deficits associated with PTSD are the least specified and understood, and the most understudied aspect of the syndrome. A major obstacle to the study of emotional response deficits in PTSD is that the concept of emotional numbing is ambiguous and poorly operationalized. For example, what is meant by 'restricted range of affect?' On its face, this symptom suggests a generalized limitation in the capacity to feel and express emotions. Such a broad definition is untenable given the intense emotional responses associated with reliving experiences in trauma [10].

If PTSD patients are restricted in some of their emotional responses, which emotions are constricted? It is not clear what precipitates or maintains emotional numbing, nor is the relationship of numbing to other symptoms of PTSD well understood. It is also not clear whether emotional numbing results directly from traumatization experiences, from premorbid characteristics (e.g. a history of coping with life-stressors by detachment), or from other post-trauma and comorbid problems such as social withdrawal.

Formally, emotional numbing is represented in three separate diagnostic criteria in the diagnostic nosology: *markedly diminished interest in significant activities* (Criterion C-4), *feelings of detachment or estrangement from others* (C-5), and *restricted range of affect* (C-6) [6]. Descriptive questionnaire studies have revealed that individuals with PTSD endorse these symptoms at a higher rate than individuals not diagnosed with PTSD [11–13]. It should be emphasized, however, that these data do not reveal very much about the process of emotion nor the parameters of emotional functioning unique to trauma and posttraumatic adjustment. For example, are the restrictions in emotion reported by patients with PTSD a problem in expressive behaviour, felt emotion, or both? Also, what triggers the activation of the so-called numbing process? A critical examination of the parameters of emotional-processing deficits in PTSD is needed to clarify these issues.

Descriptive questionnaire results are also problematic because the items tapping emotional processing deficits are typically too global and fall short in testing the limits of emotional capacities. This is particularly true if the questions posed are linked directly to the existing descriptive criteria in the DSM. However, if asked, patients with chronic PTSD are as likely to report that they choose to strategically withhold or conceal emotional reactions that they feel inside, as they are to report chronic restricted range of feelings [14]. Moreover, the external validity of reports of emotional numbing has never been evaluated. It is unclear what respondents are referring to when they report restrictions in their range of emotion, being disinterested, etc. Also it is unclear whether patients can validly self-monitor their own emotional process, particularly if the questions are global and require judgements about traits over many months, if not years (e.g. 'since the trauma'). It may be that patients' reports of their emotional problems are biased by their distress level. If so, the responses that they provide can certainly be valuable indicators of their perceived emotional difficulties, but the assessing clinician must be aware of the fact that these historical generalizations about emotional capacities may not be accurate.

Just as patients' reports may not be fully accurate, clinicians' assessments of emotional deficits in individuals with PTSD are subject to bias. Patients present for treatment when they are particularly distressed, so mental health professionals may not have the opportunity to observe the full complement of emotional behaviour of individuals seeking treatment for PTSD. In therapy, patients experience and express profound emotions that they typically seek to inhibit which may also colour clinicians' perceptions of patients' emotional repertoires. In any case, the very term 'emotional numbing' may be a disservice to individuals with PTSD because it may not accurately convey the dimensionality of their emotions. The lack of definitional clarity must be addressed if these emotional difficulties are to be adequately treated. Accurate and reliable identification of a clinical problem is a necessary precondition that must be met if it is to be successfully treated.

Theoretical accounts of emotional dysregulation in PTSD

The behavioural model

To date, the most parsimonious and widely accepted explanation of hyperreactivity to trauma-related cues in PTSD is 2-factor conditioning and instrumental learning theory. The basic learning theory account states that individuals exposed to trauma are conditioned to a myriad of previously neutral stimuli contiguous with the event, such as contextual features of a traumatic episode and concurrent thoughts and feelings [15]. Subsequently, exposure to these conditioned stimuli elicits a conditioned response that resembles the emotional reaction during the trauma (this response is termed the 'conditioned emotional response'; CER). The CER entails defensive emotional behaviour that motivates the person to escape aversive negative affect. Escape and avoidance behaviour become habitual and resistant to change. Avoidance behaviours prevent sufficient exposure to conditioned cues, which would otherwise lead to extinction of the CER. Ultimately, the conditioning theory of PTSD led directly to the use of exposure therapy, an extinction-based approach, which has been shown to be an effective intervention for trauma patients [16].

However, the behavioural model, as first proposed, does less well in accounting for the so-called emotional numbing symptoms. In the behavioural formulation, emotional numbing is seen as a consequence of the chronic avoidance of trauma reminders and reactions [15]. Findings from several studies reveal that an avoidance explanation of emotional numbing is not sufficient. Factor-analytic studies have revealed that reports of emotional numbing were distinct from avoidance symptoms [11,17]. In addition, avoidance symptoms account for a negligible proportion of the variance in reports of the three emotional numbing symptoms (disinterest, detachment, and restricted range of affect), after other clusters of PTSD symptoms are taken into account [13].

One facet of conditioning theory overlooked by researchers in trauma may account for both conditioned negative affectivity and emotional-processing deficits. The term CER was first used in a classic study of the effects of conditioning on instrumental appetitive responding in infrahumans conducted by Estes and Skinner [18]. In the original study, a tone was paired repeatedly with shocks while an animal was responding to get food. The tone itself eventually led to a decrement in the appetitive behaviour. The emotional response conditioned to the tone (CER) suppressed the appetitive behaviour. There have been many parametric studies conducted on the CER, all of which index the strength of conditioning of the CER by the degree to which it interferes with established behavioural routines (most often appetitive behaviours [19]). It has been suggested the CER suppresses appetitive motivation generally [20]. By extension, in PTSD, CERs to trauma-related cues and contexts would cause the suppression of ongoing appetitive behaviour and reduce motivation to meet appetitive goals, which would lead to emotional-processing deficits, particularly in the context of routines subsuming appetitive behaviour (e.g. affiliation, eating, sexual gratification). This extension of the learning theory of PTSD is consistent with our model (see below), in that it would predict that in the absence of conditioned stimuli, patients with PTSD would be able to perform appetitive behaviours, including the expression of positive emotion.

The biological model

Several investigators have proposed deficits of emotion in PTSD to be analogous to the sequelae observed in infrahumans exposed to inescapable shock, most notably catecholamine depletion [9], and conditioned analgesia [21]. Foa and her colleagues argued cogently that emotional numbing is a variation of the conditioned analgesia seen in infrahumans exposed to uncontrollable and unpredictable aversive stimulation [22]. These researchers suggested that emotional numbing is a phasic response cued by trauma-related contexts, akin to cued states of stress-induced analgesia brought on by exposure to contexts where uncontrollable shock was administered. This model has considerable conceptual appeal, especially in its attempt to explain how numbing-related phenomena are cued by trauma-related contexts. However, there has been little experimental research attempting to validate the model in humans, although there is some evidence of conditioned analgesia after a trauma-related challenge in PTSD [21]. In addition, the relationship between conditioned analgesia and emotional behaviour in traumatized humans remains unspecified.

Information-processing models

Horowitz proposed that trauma creates two opposing sets of internal processes, intrusion and denial, that individuals use to cope with and resolve responses to extreme stressors [7]. The 'intrusion phase' of adjustment includes the hallmark cognitive and emotional symptoms of PTSD: painful re-experiencing and hyperemotionality. Such intrusive experiences trigger an opponent process of ideational and emotional denial that represents the defensive phase of adjustment. The function of denial is to ward off painful affects and memories related to the trauma. Emotional numbing is a

component of denial, which allows patients with PTSD to minimize the feelings associated with traumatic memories. A traumatized person is said to shift back and forth between generalized unresponsiveness (numbing) and intrusion until resolution of the trauma occurs. Although Horowitz's model has had widespread heuristic value, there have been few accounts of its application in the study of chronic forms of PTSD.

A number of investigators have expanded on Horowitz's information-processing model, incorporating constructs from theories of social cognition, cognitive science, and Lang's [23] bio-informational model of fear, to explain the mechanisms that cause PTSD symptoms [24–26]. A basic tenet of these models is that cues reminiscent of the trauma trigger a neural network of trauma-related associations and action potentials that create CERs and re-experiencing phenomena. The experience of trauma is so profoundly salient and intense psychologically and biologically that over time a wide variety of internal psychological and physiological cues and external stimuli come to elicit re-experiencing symptoms predominated by negative affect. Weak or degraded trauma-relevant stimuli (e.g. ambiguous threat situations) can lead to trauma network activation in PTSD [25].

Litz [27] invoked Leventhal's [28] perceptual-motor theory of emotion and synthesized various tenets of the information-processing models of PTSD to explain emotional dysregulation in PTSD. In perceptual-motor theory, emotions are the experiential synthesis of a number of different information-processing components that can operate simultaneously: expressive-motor functions (typically facial action), learned schematic processing routines, and higher-order conceptual cognitive functions. In this perspective, unlearned expressive-motor programmes, which are the building blocks to all emotional experience [28], are not affected by stress, trauma, or posttraumatic pathology. The expressive-motor functions that were available to the individual before they were traumatized are intact, as are pretraumatic, elaborated emotional knowledge or schemas. Litz argued that the capacity to experience and express a variety of emotions is unaltered in PTSD, and the construct of emotional numbing fails to take into account the complex, dimensional, and context-dependent nature of emotional behaviour in traumatized individuals [27].

Consistent with the behavioural model, Litz argued that there is a primacy of conditioned aversive emotional reactivity and associated re-experiencing symptoms in posttraumatic adjustment [27]. Since the network of trauma memories is broadly generalized across the domains of thought, image, action, and feeling, it is more readily triggered, and therefore more accessible to experience than other emotion networks (see 24). When the trauma network is activated, other, more adaptive responses to interpersonal stimuli are less accessible and are less likely to influence behaviour [29]. Expressive behaviours associated with moderate to high pleasant feeling and other appetitive routines are expected to be less accessible to experience when PTSD patients are in states of re-experiencing, characterized by high arousal and aversive emotion cued by trauma-related contexts. In addition, when patients with PTSD are exposed to reminders that elicit CERs, they should be primed to be more responsive to emotional contexts or cues that are consistent with that state [27]. Thus, like the Foa *et al.* model [22], Litz's reformulation proposes that emotional-processing deficits in PTSD are context-dependent. However, unlike the Foa *et al.* model, Litz's model specifies differential emotional deficits associated with exposure to reminders of trauma.

Modifications to Litz's 1992 model

Since its inception, our model has evolved from our own research findings, and from the extensive normative work by Lang and his colleagues on attention, activation, and emotion [30]. We continue to assert that pretraumatic emotional repertoires are fully accessible under the right set of circumstances. However, emotional experience, particularly emotional expression, is suppressed as a result of episodes of cued CERs to trauma-related cues [31]. We have also shown that under some circumstances, as expected, PTSD is associated with hyperresponsivity to negatively valenced emotional stimuli [32].

In the revised model, we posit that trauma and the chronic re-experiencing episodes characteristic of PTSD lead also to an acquired predisposition to require more intense stimulation (higher threshold) to access the full complement of pretraumatic appetitive (positive or pleasant) emotional behaviour [33]. Regardless of context, patients with PTSD require more clear-cut and arousing stimulation to prompt a full range of positively valenced emotion. Conversely, we now also hypothesize that patients with PTSD in any context are likely to respond to negative cues with a defensive negatively valenced emotional response at a lower threshold of intensity. It bears underscoring that we do not posit that PTSD is associated with a general decline in emotional-processing capacities, like some have suggested [8]. We see no reason to assert that PTSD entails an incapacity to access any feeling state available pretraumatically.

Emotional experience and expression are a result of an intricately reciprocally interacting series of peripheral and central nervous system events (e.g. perceptual processing, orienting, the allocation of attentional resources, appraisal, the activation of expressive-motor routines,

and so forth). A number of person variables (e.g. biological capacities, learning history) and environment variables (e.g. the quality of emotional stimulation, social context) influence emotional capacities. Emotional responses are particularly constrained by the availability of cognitive resources. Patients with PTSD are prepared to efficiently and preferentially process trauma-relevant information in service of avoidance behaviour, which drains cognitive resources [24,25]. Research has shown convincingly that patients with PTSD have their attention drawn unwittingly to trauma-related stimuli in their environment [13,34,35]. We have found Vietnam combat veterans with chronic PTSD to respond as if they are preparing for threat or demand while viewing emotional images, regardless of valence [31]. We contend that the preferential allocation of attention to threat, and the arousal this process promotes, generally raises the threshold required to respond emotionally to pleasant stimuli in PTSD.

We have argued previously that adaptive emotional-processing in PTSD is hampered by reductions in cognitive resources only when the network of trauma memories is activated. We now also claim that individuals with PTSD require more intense stimulation to elicit full expressions of positive emotion, because information-processing resources are preferentially allocated to cope with threat. One useful analogy is 'signal to noise ratio', whereby the signal is the emotion eliciting stimulus and the noise is the state of the individual. In PTSD, the intensity of the signal required is higher for positive cues because the system is generally noisier. Patients with PTSD are prepotent or predisposed to respond defensively, which requires a greater background arousal in service of avoidance-motivated coping. This characteristic is inconsistent with appetitive behaviour and pleasant feelings at low to moderate levels of intensity. A system that is generally prepared to respond to threat is also primed to respond to negatively valenced stimuli at a lower threshold of intensity [36].

Our research provides some initial empirical evidence for a link between hyperarousal and emotional-processing difficulties in PTSD. After controlling for demographic characteristics, exposure to trauma, reports of depression, and each subcluster of PTSD symptoms (re-experiencing, strategic avoidance), the single best predictor of the severity of reports of emotional numbing (disinterest, detachment, and restricted range of affect) was problems with hyperarousal [13].

Empirical Support

Emotional processing in PTSD [31].

In order better to understand the link between CERs to trauma-related cues and subsequent emotional-processing deficits in PTSD, we exposed two groups of Vietnam combat veterans to an evocative trauma-related priming video (and to a neutral comparison video). Directly after presentation of the priming events, we assessed participants' emotional reactions to a set of photographic images that varied in hedonic valence (positive, neutral, and negative). Emotional responses were evaluated across a number of dimensions, including peripheral autonomic activity (heart rate, skin conductance), somatic nervous system activity, specifically indexed by facial expressive-motor response, measured electromyographically (EMG) over the zygomatic major and corrugator facial muscles, and self-reported evaluations of valence and arousal reactions.

The combat prime manipulation produced CERs in the PTSD group specifically. For example, the PTSD group reported greater negative affect and manifested higher heart rates specifically to the combat prime challenge, relative to the control group. Contrary to a global model of emotional numbing, both groups exhibited emotional behaviour modulated by stimulus valence, under the neutral condition. Consistent with our model, the PTSD group exhibited suppressed zygomatic EMG responses to positively valenced images [27], in comparison to the control group only after being exposed to a trauma-related prime. The PTSD group failed to respond to the negatively valenced images with more pronounced emotional reactions after being exposed to trauma-reminders (in comparison to the control group). This null effect requires replication. It could be that CERs to trauma cues do not serve to augment negative affect because of a ceiling effect. Interestingly, the PTSD group responded to all images, in both prime conditions, with higher heart rates, suggesting an automatic preparation for demand or threat in any uncertain emotional context. This unexpected finding supports the idea that patients with PTSD are prepotent for aversive motivation, which may lower the threshold for negative emotion generally. We are currently studying the possibility for a lower threshold mechanism for negative affect in PTSD.

Self-perception of emotion in PTSD [32].

This study was conceived as an extension of the self-perception approach to the investigation of emotional abnormalities. An examination of the relationship between expressive behaviour and subjective emotional experience in PTSD is appealing, given general agreement that PTSD is characterized, in part, by pathologies of both. Thus, our approach was to investigate the expression-experience relationship in PTSD by manipulating expressions directly and measuring consequent emotional feelings to determine the effect of the former on

the latter. We also examined the contribution of contextual cues (trauma-related *vs* neutral) to the relationship between emotional expression and experience. Consistent with Litz's model [27], we hypothesized that patients with PTSD are overly sensitive to their own negative emotional behaviour when exposed to a trauma-related context. Alternatively, when exposed to trauma cues, patients with PTSD were expected to be less responsive to expressive behaviour of positive feelings. Male Vietnam combat veterans with PTSD ($N = 20$) or with no psychiatric disorder ($N = 19$) were instructed unknowingly to adopt emotional facial expressions while listening to sounds that were either neutral or trauma-related, and after each expression they made ratings of their emotional feelings. The PTSD group reported significantly greater unpleasant feelings during both conditions, as compared with the well-adjusted group, but lesser magnitudes of happy feelings, only during the trauma condition.

The parameters of emotional responding in women with sexual assault-related PTSD [37].

We evaluated the emotional-processing consequences of script-driven imagery of personalized trauma-related narratives, relative to the effects of a personalized account of a positive event and a generic negative event in women with sexual assault histories ($n = 15$) and yoked normal controls ($n = 12$). The control group's emotional response was appropriately moderated by the valence of the narrative, whereas the PTSD group did not report statistically reliable changes in negative emotion across the conditions. This corresponded with the finding that the PTSD group failed to modulate the words used to describe negative emotion between the two conditions. However, both groups used more positive emotion words during the positive writing condition than in the negative condition. All participants showed modulated facial–motor reactions relative to the two narratives. These results suggest that negative affectivity may predominate in the self-disclosure and experience of patients with PTSD. These findings also underscore the complexity of emotional responding in PTSD and are generally inconsistent with the concept of global and pervasive 'numbing'.

Conclusion

Trauma is profoundly disruptive, emotionally. Chronic PTSD entails an admixture of intrusive negative affects and problems with emotional experience and expression, particularly positive-feeling states. In our view, the main psychological burden of trauma for patients with chronic PTSD is the ease with which memories of trauma become activated, triggering intense negative affects, which consume psychological, biological, and interpersonal resources. We hypothesize that there is a phasic relationship between these re-experiencing episodes and subsequent emotional response deficits in chronic PTSD. Our empirical research has shown that when patients with PTSD are exposed to reminders of their trauma they are constrained in their capacity to express positive feelings. Clinicians should consider the possibility that patients with chronic PTSD are able to fully access their pretraumatic positive and negative emotional repertoires or capacities, and they should generally expect that reducing the frequency and intensity of re-experiencing states in therapy should broaden emotional capacities.

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